Angiographic Pitfall: Duplicated Tapered A1 Segment of the Anterior Cerebral Artery Mimicking an Anterior Communicating Artery Aneurysm

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Summary

We describe a misleading case of a partially occluded A1 segment duplication that mimicked an ACoA aneurysm on computed tomography angiography and conventional angiography and led to surgical intervention. The location of such an anomaly at the ACoA on the side of least hemodynamic stress may provide a clue to recognizing this variant.

Introduction

Duplication of the A1 segment of the anterior cerebral artery (ACA) occurs in up to 4% of normal subjects [1-5]. Its incidence may double in patients harboring anterior communicating artery (ACoA) aneurysms [1,4,6]. This variation is frequently undetected angiographically but discovered during surgery [1,4,6]. We report a misleading case where a partially occluded A1 segment duplication mimicked an AcoA aneurysm on computed tomography angiography (CTA) and conventional angiography.

Case Report

A 70-year-old man with a medical history of hypertension, dyslipidemia, coronary artery disease, and a right middle cerebral artery (MCA)-territory stroke from a known right internal carotid artery (ICA) occlusion from which he made a full recovery presented with sudden onset headache, nausea, and nuchal rigidity. Computed tomography (CT) showed subarachnoid haemorrhage in the basal cisterns and in the interhemispheric and sylvian fissures. In addition to the complete occlusion of the right ICA, 3D-CTA revealed an image highly suggestive of an aneurysm at the ACoA adjacent to the right A1-A2 junction (Figure 1). The right anterior circulation was supplied entirely by the left A1 through the ACoA. Conventional four-vessel angiography confirmed these findings (Figure 2). Endovascular treatment of the aneurysm was deemed unsafe because the right anterior circulation was dependent on the ACoA and surgical clipping was elected. Through a left pterional approach, the anomaly suspected to be an ACoA aneurysm was found to correspond to a tapered duplication of the A1 segment of the right ACA (Figure 3). There was no evidence of aneurysm at the ACoA complex. No other aneurysm or source of bleeding could be identified despite optimal operative exposure and inspection of the entire anterior circulation bilaterally up to the left MCA and right ICA bifurcation. The majority of blood was located around the left ICA. The patient went on to develop clinical and radiographic vasospasm, which was treated with medical therapy. Repeat good quality an-

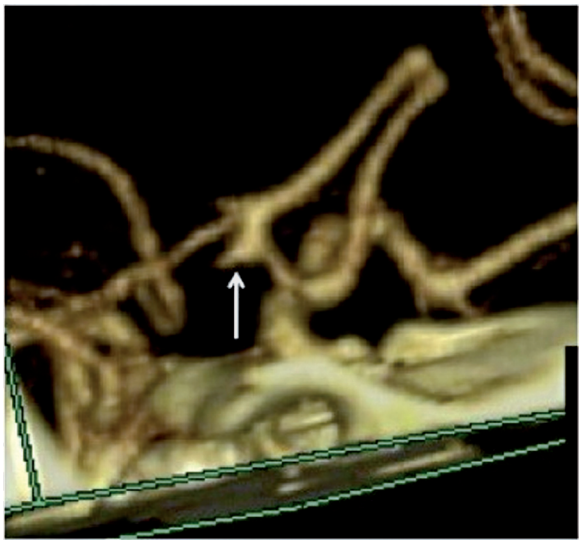

Figure 1 3D CT-angiography revealing what was thought to be an aneurysm originating from the right side of the ACoA adjacent to the R A1-A2 junction. There is complete occlusion of the right ICA and the right anterior circulation is supplied entirely by the left ICA.

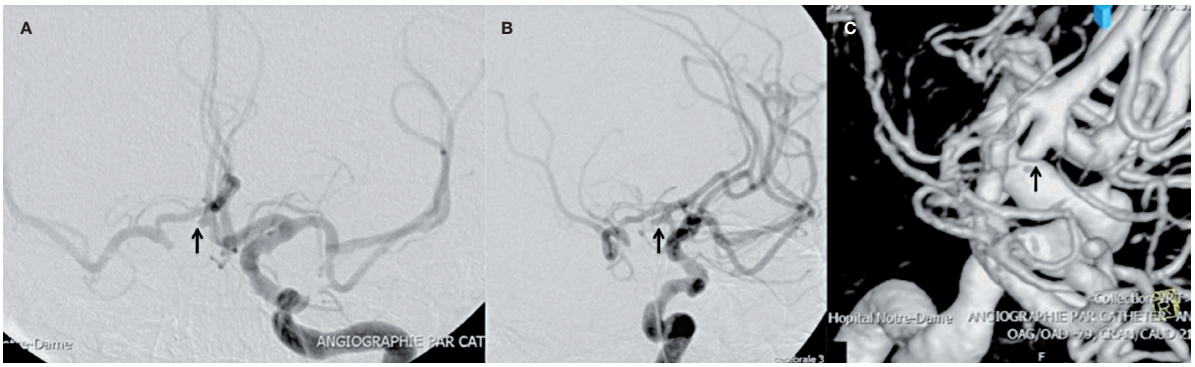

Figure 2 Conventional angiography of the left internal carotid artery (A, B) with 3D reconstructions (C) showing the supposed aneurysm originating from the ACommA complex (arrows).

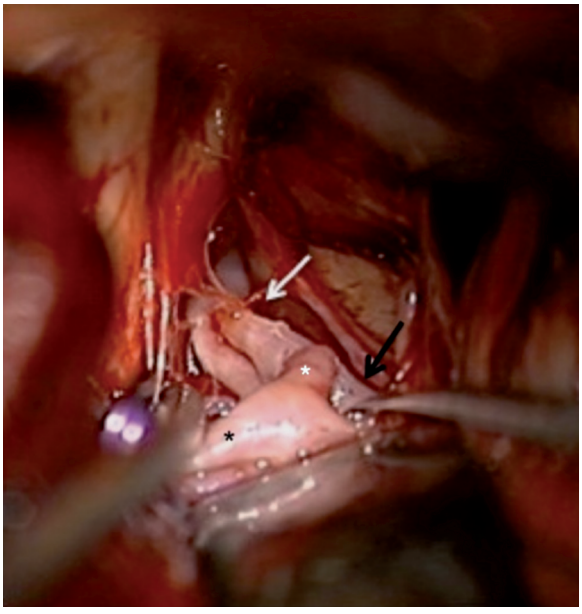

Figure 3 Intraoperative photograph through a left pterional approach showing the duplicated A1 segment of the right ACA. The proximal A1 segment (arrow) is known to be occluded on angiogram. Circulation through the A2 segment (black arrow) arises from the left A1 segment (black asterix) through the ACoA (white asterix).

giography showed no aneurysm, no significant changes, and no other possible source of haemorrhage. The patient went on to make an uneventful and full recovery.

Discussion

Anomalies of the anterior cerebral-anterior communicating-recurrent artery complex are frequently encountered [1-5], especially during ACoA aneurysm surgery [5]. Among these anatomic variations, duplication of the proximal segment (A1) of the ACA is infrequent [5]. It has been reported to occur in 4% of subjects in cadaveric studies [5] and in up to 0.5-9.7% of cases of ACoA aneurysm surgery [1,4,6]. Although A1 segment duplication can be identified on angiography [7], superimposition of vessels may render its identification difficult. Most clinically reported cases of A1 duplication are angiographically occult variations later identified during ACoA aneurysm surgery [1,4,6] usually without consequences [1,4,6,8,9]. In our case, the partially occluded A1 duplication was not recognized on preoperative CTA or angiogram as it mimicked an ACoA aneurysm. Thrombosis tends to occur at or adjacent to fenestrations and may explain the occlusion of one of the duplicated A1 segments in our patient [10]. Six previous cases of thrombosed intracranial arteries mimicking an aneurysm have been reported [11-15], including one case of a partially thrombosed fenestrated basilar artery that mimicked a vertebro-basilar junction aneurysm [11]. All but two of these reported cases, led to surgical intervention [11-15]. The decision to intervene surgically rather than embolize the supposed aneurysm was based on the risk of ACoA compromise in this patient whose anterior circulation of the right hemisphere arose exclusively from the ACoA. However, embolization of the partially occluded segment of the A1 duplication would not likely result in an ischemic event given that the occluded duplication segment is a blind pouch and no perforators were found to arise from it. Although no cause of SAH was identified, we do not believe that the thrombosed duplicated vessel was responsible for the hemorrhage. The majority of blood was located around the ICA opposite to the A1 duplication and no parietal vessel abnormalities such as dissection or blister-like changes could be identified.

Post-operative review of the angiogram in this patient revealed the atypical location of the suspected aneurysm ipsilateral to the ICA occlusion. Given that aneurysms are known to form at sites of increased blood flow and hemodynamic stress, we should have expected this ACoA aneurysm to form on the left ACoA rather than on the right side, ipsilateral to the patient's ICA occlusion [16-18]. Asymmetry of A1 is associated with aneurysm formation and is found in 58-85% of patients harboring ACoA aneurysms [19-24]. In these cases, aneurysm formation is three to five times more likely at the dominant than at the non-dominant A1-ACoA junction [20,23] and up to four times more often at the dominant A-ACoA junction than at the ACoA itself [23]. Furthermore, iatrogenic occlusion of the ICA results in an increased incidence of aneurysm formation at the contralateral side, most often at the ACoA [16-18]. To our knowledge, among the identified cases of ACoA aneurysm formation following iatrogenic occlusion of the ICA, there are no documented cases of an ACoA aneurysm forming truly ipsilateral to the occlusion [25-39]. Therefore, if the suspected image in our patient was in fact an aneurysm, one would have expected it to be found on the ACoA contralateral to the side of his ICA occlusion or broadly based on the ACoA itself. However, in our minds, this argument is not strong enough to prevent surgical intervention.

Conclusion

A1 segment duplication with one arm occluded, can mimic an ACoA aneurysm on angiography. The location of the anomaly at the ACoA on the side of least hemodynamic stress may provide a clue to suspect this variant.

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